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Authors' Response

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Dear Editor,

We thank Dr. Finsterer and Dr. Stollberger for their interest in our published manuscript, which focuses on the collected data from patients hospitalized in our multidisciplinary COVID-19 department who experienced Tako-Tsubo syndrome (TTS) during the second and third waves of the pandemic [1]. This response represents an opportunity to highlight some limitations of the study and to further discuss the cases we have presented in the paper.

As regards the potential triggers of TTS in our cases, although it is not possible to establish with certainty the specific cause of TTS, it is worth noticing that all our patients do not experience only a COVID-19 infection but a full-blown COVID-19 with the known correlated symptoms and related interstitial pneumonia confirmed by computed tomography, which can represent in itself a trigger like several other infectious diseases. In addition, although, as specified by Finsterer et al., psychiatric and neurologic disorders may be triggers of TTS, the majority of TTS cases associated with neurological disorders that are reported in the literature are described after a stroke or subarachnoid hemorrhage or seizure [2]. Moreover, the literature's cases of TTS associated with Alzheimer's disease are anecdotal. For this reason, it is difficult to consider Alzheimer's disease a classical trigger for TTS. Nevertheless, we reported this information in the manuscript in order to highlight the association, which can also be interpreted as an additive predisposing condition to TTS. Larger clinical records are necessary to better clarify this interesting association. Although we recognize that anxiety may be a further potential trigger underlying the pathophysiology of TTS in these patients, the mechanisms of TTS in COVID-19 patients remain unclear and might be multifactorial.

Case #2 with atrial fibrillation (AF) was a case of permanent AF with a normal ventricular response and no symptoms. It is unlikely that a chronic and stable condition can represent in itself a trigger for TTS. However, we think that AF, in particular the paroxysmal form reported for patient #4, may be considered a complication of TTS.

For what concerns the patients with a history of colorectal cancer, the diagnosis was dated 3 years before and it was treated only with surgery at that time. Following that, the patient underwent serial oncological controls that resulted negative. As for the case of permanent AF, it is unlikely that a 3-year-dated diagnosis of colorectal cancer can be considered a trigger of TTS.

Regarding patient #4, a coronary artery fistula (CAF) is diagnosed in 0.2-0.4% of patients undergoing coronary angiography. Moreover, a recent paper collected clinical data from 9 patients with concomitant TTS and CAF, concluding that the coexistence of TTS and CAF may be considered coincidental [3]. We agree with the colleagues that a limitation of the study is the missed coronary angiography for patient #2, but our choice was forced by an intercurrent episode of hemorrhagic shock due to a spontaneous psoas muscle hematoma, which required red blood cell transfusions. Furthermore, we want to point out that several limitations of our study must be interpreted in light of the pandemic context in which we were forced to work.

With the present letter's response, we wish to make sure that our paper is more comprehensive. In conclusion, we would like to think of our work as a small piece of a wider puzzle, which is the pathophysiology of TTS in COVID-19, which remains still to be defined. Further and larger studies will certainly help to better understand this interesting association.

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